Environmental Effects

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Trends in Public Health in the Population Near Nuclear Facilities: A Critical Assessment

By C. H. Patrick*

Abstract: Ten studies that have looked specifically at changes in public health in areas near nuclear facilities are critically reviewed. All but one of these studies have been unable to show adverse health effects in the local population that might be related to radiation exposure. The one study that purports to find an adverse effect has severe methodological limitations, which preclude any meaningful interpretation of the data.

Also presented is an analysis of the indicators of public tealth in the area of Oak Ridge, Tenn., which shows cancer mortality rates that are not significantly higher than would be expected in the general U. S. population.

Although much more research is needed before all the effects of very low levels of radiation from nuclear reactors will be known, the existing studies suggest that nuclear power plants will not have a significant impact on public health as a result of normal operations.

health hazards from very low levels of radioactivity from releases made during normal operations of nuclear facilities. This is due in part to the lack of knowledge of the effects on man of exposure to very low levels of radiation. However, if the consequences of low-level releases from a nuclear facility are deleterious, then increases in measures of ill health associated with radiation exposure should be observed in the population living near the facility as compared to a control population. 9-1 1

There are numerous conflicting reports concerning the

To properly study the public health effects of nuclear facility operations, one must have data on relevant measures of ill health for the nearby geographic areas for periods both before and after the facility begins operations. A preliminary analysis of vital statistics data† (such as deaths, illnesses, births, and population sizes), adjusted for demographic variables (such as age, race, and sex) that reveal significant

^{*}Clifford H. Patrick is a member of the Human Health Studies Program, Division of Biomedical and Environmental Research, Energy Research and Development Administration (ERDA). He is currently on leave of absence from Oak Ridge National Laboratory, where he has been a member of the research staff since 1971. He received the Ph.D. degree in economics (with a specialization in demography) from Duke University in 1971. He was a Postdoctoral Fellow in Epidemiology at Johns Hopkins University during the 1975 to 1976 academic year. In 1974 he was codirector of occupational safety and health training programs in the College of Industrial Management and Textile Science at Clemson University. He has been principal investigator of an ERDA-funded study to determine the adequacy of data and methods for examining the impact of power-plant operations on the health of the general population. His fields of interest are occupational health and safety and the regional, social, economic, and public health impacts of energy-production systems.

[†]Vital statistics data are generally published annually by each state for counties and large cities in a vital statistics series. The data are usually broken down by race, but seldom by other traits.¹² Vital statistics data seldom contain migration data and usually contain morbidity information only on communicable diseases. However, the Bureau of the Census publishes estimates of population change and migration and some morbidity data, which are available through the U. S. Public Health Service.¹³⁻¹⁵ The federal government also publishes annual vital statistics and related demographic data through the National Center for Health Statistics and through the Bureau of the Census.¹⁴.¹⁷

time and geographical trends apparently related to nuclear power-plant operations leads to additional analyses, including the social and economic structure of the population, which must be considered. These additional analyses could include, for example, the abrupt shifts in the socioeconomic composition of the local population due to site construction. 18.19 If the trends of ill health apparently related to power-plant operations persist after correcting for demographic and socioeconomic factors, then, whenever possible, records of radioactive releases into the environment must be obtained, dose to the population must be estimated, and ill health must be correlated with dose.20 All studies to date that have made these analyses have found no significant trends of ill health related to nuclear power-plant operations. 21.22

The purpose of this article is to critically review the studies of trends in public health in areas near several nuclear facilities, some of which have been operating since the mid-1940s. In addition, three topics of research are briefly reviewed: (1) changes in measures of ill health which are related to radiation exposure of the population surrounding nuclear plants, (2) the problems in using vital statistics for such studies, and (3) the types of analyses needed in research and in environmental impact statements.

The somatic and genetic effects that are believed to be associated with radiation exposure are well documented.23-28 Both types of effects are of interest to the epidemiologist trying to determine the effects of low-level radiation releases on public health. Clinically, many health effects induced by radiation are little different from those induced by other causative agents.29 Therefore the presence of a health effect does not ensure that the causative agent has been correctly identified. On the other hand, the absence of the hypothesized effect when the agent is present often is taken to indicate that causation has been disproved. To truly test the hypothesis, one must estimate the expected size of the effect and determine if the population and methodology are adequate to detect the expected effect.

The possible somatic effects of radiation include various types of cancers, most of which have relatively long latent periods. The cancers most often cited as caused by radiation exposure are leukemia and cancers of the thyroid, bone, breast, lung, and gastrointestinal tract. The noncarcinogenic diseases associated with radiation exposure include cataracts, central-nervous-system disorders, premature aging ("life shortening"), fertility impairment, congenital defects, and cardio-vascular renal diseases.

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The possible genetic effects of radiation exposure include gene or point mutations and chromosomal aberrations, which may produce increased rates of spontaneous abortion or fetal wastage, neonatal and infant mortality, infertility, and congenital malformations.

The human health effects associated with nuclear facilities have been examined in a wide variety of studies (see Table 1). These studies generally fall into one of two categories, both of which are reviewed in this article. Those in the first category analyze vital statistics for the area near a potential source of radiation exposure, usually a nuclear power plant. These studies look for changes in selected vital statistics of local population groups compared to population groups that are not near the nuclear power plant, and they usually look for a dose-dependent effect. Often the vital statistics before and after the facility starts operation are compared. Studies in the second category compare the vital statistics of the work force in a nuclear facility with the vital statistics of the general population. Mortalities of radiation workers and nonradiation workers have occasionally been compared. In addition, an analysis of mortality in the Oak Ridge, Tenn., area is presented.

SELECTED VITAL STATISTICS OF POPULATIONS NEAR NUCLEAR FACILITIES

The work of Bailar and Young is an example of the first category of study.30 Their research was undertaken because of a concern over the possible adverse health effects of low-level radioactive releases from the Hanford nuclear reservation near Richland, Wash., which had been raised in an earlier study by Fadeley 3 1 Bailar and Young corrected several basic errors in the Fadeley study and specifically tested the hypothesis that the higher observed incidence of cancer mortality (leukemia was considered separately) was related to the presence of the Hanford facilities. The basic errors in the Fadeley study are as follows: (1) several counties in the geographic area being studied were omitted without explanation; (2) basic data (numbers of deaths) were not reported, and statistical variations of rates calculated on small samples were not considered; (3) rates were not adjusted for age or sex even though population structures of the counties varied; (4) urban -rural variations in cancer rates were not considered; and (5) cancer mortality prior to operation of the nuclear facilities was not analyzed.30

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Table 1 Summary Review of Studies of Nuclear Facilities

Study	Nuclear facility	Year(s)	Measures of health effect	Findings and comments
Bailar and Young (Ref. 30)	Hanford Wash. (plutonium production plant; miscellaneous research facilities)	1934-1963	County vital statistics; total cancer rates; leukemia rates	No effect found; control areas employed; standard- ization used; before and after analysis; migration not considered; latency a potential problem
Tompkins et al. (Ref. 32)	Humboldt Bay Power Plant (boiling-water reactor) Dresden Nuclear Power Station (boiling-water reactor) Big Rock Point Nuclear Plant (boiling-water reactor)	1958-1962; 1964-1967 1955-1959; 1961-1965 1957-1961; 1963-1967	County vital statistics; infant mortality and rates; neonatal mortality and rates	No effect found; directional quadrants used; regression analysis used but not reported; no racial adjustment
DeGroot (Ref. 33)	Dresden Nuclear Power Station (boiling-water reactor) Shippingport Atomic Power Station (pressurized-water reactor) Indian Point Station (pres- surized-water reactor) Brookhaven National Lab- oratory (research reactors; miscellaneous facilities)	1950–1967 1950–1967 1950–1967 1951–1968	County vital statistics: infant mortality rates	Linear regression only; overall no effect statisti- cally; one positive result (Brookhaven), one nega- tive result (Shippingport) reported; independent variables: time, radioac- tive discharges, infant mortality in control areas; no racial adjust- ments; R ² (coefficient of determination) not re- ported
Sternglass (Ref. 34)	Hanford, Wash. (plutonium production plant; miscellaneous research facilities) Dresden Nuclear Power Station (boiling-water reactor) Big Rock Point Nuclear Plant (boiling-water reactor) Humboldt Bay Power Plant (boiling-water reactor) Nuclear Fuel Services, Cattaraugus, N. Y. (fuelreprocessing plant) Peach Bottom, Atomic Power Station (gas-cooled reactor) Indian Point Station (pressurized-water reactor)	1940–1945; 1946–1949 1955–1968 1962–1968 1958–1969 1960–1968 1962–1969 1958–1969	State and county vital statistics; state and county infant mortality; premature birth rates; leukemia rates	Author interprets each analysis as showing a positive effect; several errors in data presented in tables and figures; no demographic adjustments; questionable interpretation of data, often only two points; weak statistical analyses; omissions and selective inclusions never justified; dubious use of states as units of analysis; see text for further comments
	Brookhaven National Lab- oratory, Upton, N. Y. (re- search reactors; miscel- laneous facilities)	1955-1967		

(Table continues on the next page.)

Table 1 (Continued)

		Table I (Con	inuea)	······································
Study	Nuclear facility	Year(s)	Measures of effect	Findings and comments
Tokuhata et al. (Ref. 35)	Shippingport Atomic Power Station (pressurized-water reactor)		County vital statistics; total and selected cancer rates, fetal deaths, infant deaths, neonatal deaths	No effects found attribu- table to radiation; demo- graphic adjustments; migration considered; matched communities; fairly thorough discussion of needed adjustments in use of vital statistics; good methodological sec- tion
Grahn (Ref. 36)	Big Rock Point Nuclear Plant (boiling-water reactor)	1950-1971	County vital statistics; in- fant mortality; immature births; cancer mortality	No effects found; male and female rates analyzed; local area rates compared to state rates; demo- graphic factors considered
Moshman and Holland (Ref. 39)	Oak Ridge, Tenn. (gaseous diffusion plant; uranium processing plant; research reactors; miscel- laneous facilities)	1948	Cancer morbidity	Significantly lower cancer morbidity in Oak Ridge compared to nation; latency a potential problem; only study of morbidity, limited to 1 year; apparently the first study of population near a nuclear plant
Mason et al. (Ref. 37)	Grand Junction, Colo. (uranium mill tailings)	1950-1971	National Cancer Institute county data; cancer death rates; lung cancer; leukemia	No trend found attributable to mill tailings; demo- graphic adjustments; comparison with other parts of Colorado; latency problem
Larson et al (Ref. 40)	Oak Ridge; Tenn. (see above)	1950-1971	Actual deaths vs. expected deaths from all causes using man-years	Found 692 deaths, while 992 were expected, based on U. S. 1962 rates man- years analysis; problem of comparability of population
Scott et al. (Ref. 41)	Oak Ridge, Tenn. (see above)	1951 –1969	Actual deaths vs. expected deaths; man-years analysis	Uranium workers have lower mortality than non-uranium workers; U. S. 1962 life tables used for relative comparison; demographic adjustments made; death information from Social Security possibly incomplete for either group; uranium group 5 years older on the average, with more males

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Bailar and Young analyzed county data from Oregon and Washington and data from the U.S. Bureau of Vital Statistics for groups of counties near or downstream from Hanford in both states for the years 1934 to 1963. These data were corrected for differences in cause-of-death classifications and standardized for age and sex by an indirect method using the 1950 U.S. white population.

In terms of the hypothesis tested, the findings are suite interesting. Although the total cancer mortality ates in the counties of Oregon and Washington that were studied have been consistently lower than in the Jnited States as a whole, the leukemia rates in these areas have been consistently higher. Moreover, these higher death rates from leukemia have persisted since the mid-1930s, a decade before the Hanford nuclear acilities existed. In addition, leukemia rates in the river counties," including Hanford and downstream areas, have actually decreased since 1950, reversing an earlier upward trend.

Bailar and Young conclude, "No evidence was found that persons living downstream from the Hanford reservation or along the Pacific coast of Oregon had had an excess risk of death from cancer in general or leukemia in particular." It can be argued that migration has not been considered and that less than 20 years of data from the beginning of Hanford operations is insufficient to allow for discernible "excess" cases because of long latency periods. Yet, given the data available at the time and the comparison of the pre- and postoperational periods, the research suggests that no apparent carcinogenic effect over the period was due to the Hanford operations.

In 1970 Tompkins et al.,32 DeGroot,33 and Sternglass34 published studies that set out to determine if any adverse health effects on infants in utero were caused by the operation of nuclear power plants. None of the three studies appear to have standardized for maternal age or race. Tompkins et al. examined infant and neonatal mortality rates for the 5 years before and after start of operations at the Humboldt Bay (Eureka, Calif.), Dresden (Morris, Ill.), and Big Rock Point (Big Rock Point, Mich.) power stations.32 This study was undertaken in response to claims that nuclear power stations expose surrounding populations to radiation which, even at low levels, results in increased infant mortality. For a geographical distribution to be established, county infant mortality data from vital statistics were determined for four concentric bands extending a total of 200 miles from the facility. These bands were then divided into quadrants to allow prevailing wind directions to be taken into consideration. Data from the 1960 U. S. Census were used to determine population, live births, and deaths in each quadrant. Infant mortality rates were based on 5-year aggregates to reduce statistical fluctuations due to small sample size.

No relation between the operations of any of the three plants and changes in infant and neonatal mortality rates was found in the Tompkins et al. analysis. The authors further checked their results, via regression analysis, for sensitivity to either the band width or compass direction from the plants. In both these latter tests, no statistically significant relation was found. This study differs from the Bailar—Young study in (1) its measure of ill health, (2) the use of quadrants of concentric bands, and (3) the use of shorter time periods. Nonetheless, neither study finds a relation between changes in their measures of ill health and the operation of local nuclear facilities.

DeGroot's study³³ of the relation between trends in infant mortality and effluent releases from four nuclear reactors utilizes regression analysis solely. The four reactors he studied were the Dresden reactor (Morris, Ill.), the Shippingport reactor (Shippingport, Pa.), the Indian Point reactor (Indian Point, N. Y.), and the Brookhaven reactor (Upton, N. Y.). He examined the relation using a regression model of the form $M_t = B_0 + B_i X_i$, where M_t is the annual infant mortality rate for a given county of study for the years 1950 to 1967. The independent variables he used vary from case to case and include (1) the year, (2) a measure of liquid discharges, (3) annual infant mortality rates for a control area (state, nation, or other county groups), (4) gaseous discharges, and (5) background radiation (for the Brookhaven reactor).

Although DeGroot fits both linear and semilog models, his results are essentially unchanged by the log transformation of the dependent variable. Unfortunately, he does not report all the slope coefficients in his equations, and so it is difficult to assess how well the independent variables "explain" the trends in infant mortality rates. However, he does report the "t" statistics for each variable. The annual infant mortality rates and the measures of radiation effluents he used, in all but two cases, are not statistically related at the 95% confidence level. In the two cases in which there is a statistical relation, one was small and positive and one was small and negative.

In the analysis of Suffolk County mortality rates, DeGroot finds a statistically positive correlation (b coefficient of ± 0.015 , t value of ± 4.17) between the annual infant mortality rates and the 2-year moving average of tritium discharges from the sand filter beds

of the Brookhaven reactor. On the other hand, in the analysis of mortality rates for Allegheny County, Pa., which is southwest of the Shippingport reactor, he finds a negative effect (b coefficient of -0.021, t value of -2.60). DeGroot states that the differing statistical signs illustrate his contention "that it is not possible to derive strong conclusions about either the existence or nonexistence of an effect from the simple regression models..."

DeGroot's paper emphasizes the limited value derived from testing hypotheses using vital statistics for a large area in conjunction with a single point source of an effluent. Although this method may point out areas for hypothesis testing using more precise epidemiological and statistical methods, it can never be sufficient to prove or disprove a hypothesis because of the inevitable violations of the assumptions of the linear regression method.

Only one author (Sternglass) found a relation between infant mortality and low-level-radiation releases.34 Sternglass examined the vital statistics for selected years and selected areas and found a rise in infant mortality near nuclear power plants. For the Hanford facilities, several examples showing a positive relation were presented. First, he compared state data on percent change in infant mortality from 1946 to 1949 to the least-squares-fitted trend from 1940 to 1945. Whereas Washington and Oregon showed negative changes (declines) in each year from 1946 to 1949, Sternglass interprets positive increases in infant mortality in Montana and North Dakota for all 4 years and in Idaho for 2 of the 4 years as indicating a positive effect due to Hanford's operations in Washington. He further "confirms" his interpretation of the 4-year data by presenting a bar graph of the percent increase of infant mortality (not mortality rates) in 1945 over the 1943 level-before and after Hanford went into operation—for counties surrounding Hanford and for other distant control counties. He does not note that this increase in the number of infant deaths is related to the population growth in the area as a result of the construction of the Hanford facilities.

Sternglass also studies mortality rates for the areas near the Dresden reactor in Illinois. He finds the infant death rate in Illinois during 1959 to 1968 is consistently higher than that of Ohio. Even though the rates in Illinois were decreasing over the previous year's rates (except during the Illinois rubella epidemic of 1964 and 1965), the difference in the states' rates is correlated (0.865) with radioactive releases from Dresden. He also cites comparisons of Illinois infant mortality with that of North Dakota, Indiana, and

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Michigan but fails to account for the overall decline in these rates or for demographic differences among the populations.

Sternglass then compares infant mortality rates for 1964 with those for 1966 in six counties surrounding Dresden (including Will County in the Chicago metropolitan area and Grundy County in which Dresden is located) and makes the same comparison in six noncontiguous counties in northern and western Illinois. The infant death rate in the six counties around Dresden increased from 20.8 to 24.3 per 1000 live births, whereas the rates in the six control counties rose from 22.9 to 23.3 in the 2 years. Neither a standardization for demographic variables nor a comparison of data for other years is cited.

Sternglass also compares premature birth rates in his six control counties with those in Grundy County alone. Grundy, with an estimated 1964 population of 23,500, had premature birth rates for 1964 to 1968 of 3.6, 6.3, 8.7, 7.2, and 5.0%, respectively. Among his control counties, the smallest of which had a 1964 estimated population of 39,500, the lowest rates for those years were 5.5, 4.6, 5.1, 5.2, and 6.1% and the highest were 7.5, 5.9, 8.2, 7.3, and 7.7%, respectively. Sternglass cites the large rise in Grundy County in 1966 (the year of peak emissions) as evidence of the adverse effect of the Dresden reactor. However, his control counties also showed evidence of such a peak, which he does not explain.

Sternglass uses similar interpretations of limited data to show that infant mortality has risen in the vicinity of Humboldt, Calif.; Cattaraugus, Westchester, and Suffolk, N. Y.; and York, Pa., as a "result" of radioactive releases, and again he fails to take into account normal statistical fluctuations and other factors associated with differential infant mortality rates.

The Sternglass paper is discussed in detail here because of his gross errors in using vital statistics, not because he claims to see an "effect" due to nuclear power reactors. The paper illustrates a number of methodological pitfalls in using vital statistics and limited quantitative analysis. He adeptly chooses isolated data from selected years and locations and uses various "analyses"; he uses no consistent methodology, nor does he standardize the rates to account for real differentials due to population characteristics. Yet he unfailingly interprets the outcome to show an adverse effect of radiation when the data are inadequate to support such an interpretation. Such studies do little to clarify the true relation between changes in public health and exposure to low levels of radiation.

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Two additional studies, by Tokuhata et al.35 and by Grahn, 36 have examined the public health impact of nuclear facilities. These studies were undertaken in response to claims of increased rates of mortality due to releases from these plants. In 1974, Tokuhata et al. published a study analyzing health hazards to the public living near the Shippingport nuclear reactor in Shippingport, Pa.35 Using vital statistics and census lata for 1961 to 1971 for Aliquippa and communities f similar demographic background without nuclear ower plants, they examined fetal and infant mortality ates and those from leukemia and other neoplasms. Additional analyses were also made to determine if geographically distributed radiation-dose-related effects were present. Mortality rates at 5-mile intervals from the reactor and the differences in mortality rates for "on-river" and "off-river" communities downstream from Shippingport were examined.

On the basis of their comprehensive analysis, Tokuhata et al. concluded that "there is no systematic evidence to support the allegation that radioactive releases from the Shippingport plant have had significant effects on the health of the population in the vicinity of the plant..." that cannot be explained, at least to some extent, by reporting errors or other known sociological characteristics of the population.

Although the Tokuhata results confirm those of earlier studies, perhaps the major value of the paper lies in its discussion of the problems associated with analyses of this genre, especially the shortcomings of published vital statistics. The paper clearly points out sources of potential error and, more importantly, errors in the public health data. It also attempts to correct for these shortcomings where possible. For example, black infant mortality rates were consistently higher, by a factor of 2, than white rates. Therefore communities that have had a high recent influx of black families or have a relatively high proportion of blacks, as Aliquippa does, compared to the state population will have higher rates of infant mortality. Further analysis by race is then necessary to correct for this source of bias.

Grahn presents another analysis of the Big Rock Point nuclear plant in Michigan. In addition to infant mortality, which was also examined by Tompkins et al., Grahn analyzes cancer mortality and premature birth rates for eight counties surrounding Big Rock Point. The decade prior to 1962, before Big Rock Point began operations, and the following decade ending in 1971 are included in the analysis. In addition, Grahn considers changes since 1950 in the socioeconomic and demographic composition of the

population, both in absolute terms and relative to Michigan as a whole.

Overall, Grahn finds no evidence to indicate that releases from the Big Rock Point nuclear station increase ill health in the surrounding population. Specifically, he finds that (1) the rate of premature births is equal to or below the state mean; (2) infant mortality has been above the state averages for the past 20 years, including a decade prior to the reactor startup, but has been declining in recent years; (3) cancer death rates in the area are below state averages, and, for women, have been declining, especially in Charlevoix County where the nuclear power plant is located; and (4) leukemia rates are lower than the state average for females and are about the same as the state rates for males.

Because of concern over the use of uranium mill tailings as construction fill material in western Colorado, a study of the counties surrounding Grand Junction, Colo., was conducted by Mason et al. to determine if higher than normal cancer rates were discernible.³⁷ Mason et al. examined the age-adjusted cancer mortality rates for white males and females from 1951 to 1967 for leukemia, lung cancer, and all other cancers compared to cancer mortality rates for the 1960 Colorado white population.

In comparing the data for the counties, the investigators could find no carcinogenic effect due to radiation exposure from the mill tailings. Cancer mortality rates for females and for males under 20 years of age showed no statistical difference from the rates in other counties. Leukemia mortality rates for males were no different from those in other counties, but the male mortality rates for lung cancer and for other cancers were higher than those in other counties in the state. However, these rates for males in the one county where tailings were used extensively in construction were consistently below those of counties where tailings were not used. The authors correctly point out that latent periods of 15 years or more may be involved and, if so, that the effects would not yet be observed in the data. Therefore the evidence remains of limited value until it is possible to extend the study over the longer period. In such studies population migration also may be a serious complicating factor, as are personal factors such as smoking history.

Moshman and Holland³⁸ have studied the population near Oak Ridge, Tenn., although only for the year 1948. Moshman and Holland compared the incidence of cancer morbidity in the Oak Ridge population with the expected incidence to determine if Oak Ridge residents were more susceptible to cancer than the

general population. They computed age-standardized cancer incidence rates, based on the 1940 U.S. population age structure by primary site and total cancers for males and females. They found that cancer incidence in Oak Ridge was only 123 per 100,000 compared to the national average for whites of 230 (and the death rate from heart disease was 46 per 100,000 compared to the national rate of 320), reflecting the healthy, highly selected Oak Ridge population. Incidence rates for both males and females were lower than the national norms. On a relative basis the distribution of primary cancer sites in white females in Oak Ridge was not significantly different from the nationally observed distribution. A higher proportion of respiratory cancer was found in white males than would have been expected. The authors feel this was due to the increase in lung-cancer rates over the decades since the 1940 population was analyzed.

Overall, this study of Oak Ridge cancer morbidity is rather a limited use of vital statistics, but it must be considered in perspective. It appears to have been the first study recognizing that nuclear facilities may be potential sources of ill health, and the study set out to test this hypothesis. To my knowledge, it is still the only study using morbidity, or illness, rates as opposed to the more readily available death rates. Given these conditions and recognizing its limitations, the paper is indeed a valuable contribution to the overall picture being formed by studies of vital statistics in areas containing nuclear facilities.

COMPARISON OF VITAL STATISTICS FOR NUCLEAR FACILITY WORKERS AND FOR THE GENERAL POPULATION

As previously noted, the second category of study of the health effects near nuclear facilities compares the vital statistics of the work force in a nuclear facility with the vital statistics of the general population. This type of study was undertaken by Larson et al.39 and Scott et al.40 Both studies attempted to determine if working in the Oak Ridge facilities increases the risk of mortality for the employee. The 1966 study by Larson et al. compares the number of employee deaths in the three Oak Ridge nuclear plants from 1950 to 1965 with the number of deaths expected by applying 1962 U. S. sex- and age-specific white mortality rates to the age and sex distribution of the workers. 39 On the basis of 207,204 man-years of cumulative employment, 992 deaths would have been expected, but only 692 deaths had occurred by the end of 1965. Thus workers

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exposed to the environment of a nuclear facility appear in this analysis to live longer than persons in the general population. This discrepancy probably can be explained by the mortality contributions of institutionalized and unemployable persons in the general U. S. population whose health is poorer than that of workers, such as those at the Oak Ridge facilities, who have on-site medical care and periodic plant physicals.

Scott et al.40 divided workers from two of the Oak Ridge facilities into two groups, uranium workers and nonuranium workers, based on their work areas at the plants. The uranium workers were predominantly technicians and craftsmen, whereas the nonuranium workers covered a broader spectrum of job classifications. The study covers the years 1951 to 1969 and applies the 1960 U.S. mortality tables to each of the two distributions to determine expected deaths in each group. Scott et al. found the mortality rate for uranium workers to be only 59% as high as the 1960 U.S. control population, whereas the mortality rate for the nonuranium workers was 76% as high. Thus the uranium workers had a lower mortality rate than the nonuranium workers. This result may be even more significant because the average age of the uranium workers is about 5 years greater than that of the nonuranium workers. This potentially gives radiation a longer time to cause an adverse effect if both groups were hired at the same age. Also, the nonuranium worker group contains eight times more female workers, who have lower mortality rates than do males (although the analysis is sex adjusted).

Although there is little doubt that the mortality rate for uranium workers is lower than expected, two critical questions are left unanswered by this study: (1) What are the causes of death, and how do the rates for these causes differ from those in the mortality tables for the general population? (2) How does uranium worker mortality change with radiation dose? These questions should be answered by the Energy Research and Development Administration's (ERDA's) health and mortality study of workers from which only preliminary analyses have appeared to date.⁴¹

PRELIMINARY ANALYSIS OF MORTALITY IN THE OAK RIDGE, TENN., AREA

Trends in four measures of mortality for the Oak Ridge, Tenn., area from 1929 to 1971 are examined in this section. Included are a 14-year period prior to the existence of the city of Oak Ridge and its three nuclear

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ity for the Oak :amined in ioa prior to the ts three nuclear facilities and the subsequent 29-year period.42 The analysis has been restricted to the white population for three reasons: (1) the nonwhite population is usually quite small, generally younger, and subject to much larger errors in reporting, especially prior to the 1950s, than is the white population; (2) nonwhite rates of age-specific mortality by cause are approximately twice cose of whites; and (3) an effect from a nuclear power :nt should show up equally among racial groups.

The crude vital statistics from the city of Oak lige were compared with those from the state of nnessee in an attempt to evaluate the relative shifts the incidence of mortality from various diseases, nich might suggest a hypothesis concerning an effect radiation.

Fetal and infant deaths and deaths from congenital alformations have been slowly declining in the white pulation of Oak Ridge and the state of Tennessee. owever, cancer in these same populations has been acreasing. In Tables 2 to 4, the actual number of caths from selected causes, the population size, and he death rates are shown.

If only the period from 1949 (when the first vital tatistics for Oak Ridge became available) to 1971 is examined, as in Figs. 1 and 2, the trends in deaths for the four causes of death reflect no particular sequence which would suggest that the Oak Ridge area has been or is becoming a relatively hazardous locale. Since the number of deaths is small, the statistical fluctuations could be large, but the trends are fairly consistent. The city of Oak Ridge, which is closest to the nuclear facilities, does not show any consistent increases, nor do Anderson County and Roane County. All three areas reflect the general mortality trend indicated for the state of Tennessee.

Although crude annual rates are frequently used, as discussed previously, age-adjusted annual rates are the only appropriate data for comparing the city and state death rates. However, age-adjusted rates are not published for the state of Tennessee. Even those rates which are published may be erroneous. For example, misreporting of a few deaths could introduce a large bias into the smaller reported figures, and misreporting is likely because of changes in death classifications every decade.

A second source of error is obvious in the crude death rates for the Oak Ridge area. The crude death rates are based on the ratio of the number of deaths to the estimated size of the population. As shown in Table 2, these population estimates are unquestionably inaccurate for many, if not most, intercensal years. For example, between 1943 and 1949 Anderson County's base population, upon which the rates are based, did not include Oak Ridge, but the mortalities did. For these reasons the data are only plotted and discussed but not analyzed statistically.

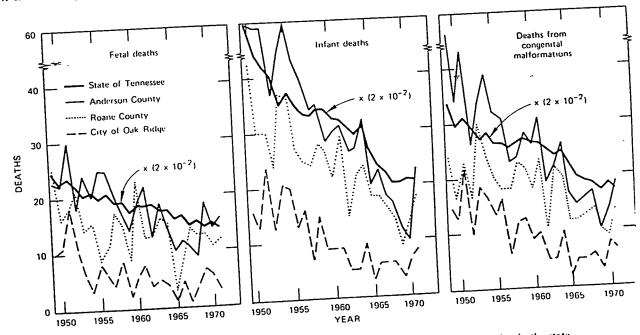


Fig. 1 Trends in fetal, infant, and congenital malformation deaths in the white population in the state of Tennessee and in the Oak Ridge area, 1949-1971. Source: Ref. 42.

Table 2 Deaths from Selected Causes in the White Population Proximate to Oak Ridge, Tenn., 1929-1971ab

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		Anderson County ^c Roane County ^c								•	City of	Oak Ri	dge ^C		
Year	Pop.d	(1)	(2)	(3)	(4)	Pop.d	(1)	(2)	(3)	(4)	Pop.d	(1)	(2)	(3)	(4)
1929	18,971	24	25	7	9	22,571	19	50	12	16					
1930	19,283	15	32	5	15	23,024	29	48	12	17					
1931	19,418	13	31	4	16	23,024	21	31	16	12					
1932	19,554	22	35	1	12	23,024	19	52	11	19					
1933 ^d	19,689	21	44	12	24	23,024	20	39	13	14					
1934	19,825	32	31	5	16	23,024	19	27	11	9					
1935	19,960	26	45	9	10	23,024	11	41	20	15					
1936	20,096	22	51	11	21	23,024	11	49	14	18					
1937	20,232	18	35	8	18	23,024	12	47	18	18					
1938	20,367	19	32	14	14	23,024	24	46	11	16					
1939	20,503	8	24	12	11	23,024	13	24	14	7					
1940	26,176	10	29	11	19	26,471	17	41	24	13					
1941	26,851	10	42	9	28	26,807	10	26	9	11					
1942	27,526	2	33	8	13	27,144	9	36	17	12					
1943 ^e	28,201	14	26	9	8	27,480	14	28	17	11		Oak R	idge bu	ilt	
1944	28,876	15	45	12	33	27,816	17	53	25	25					
1945	29,551	23	71	23	43	28,153	23	53	13	22					
1946	30,226	24	64	39	46	28,489	21	43	19	24					
1947	30,901	43	65	30	49	28,825	23	33	29	23	N	o data i	before	1949	
1948	31,576	40	58	35	46	29,161	16	38	32	25					
1949	54,997	23	61	36	46 ^f	29,852	25	42	22	24 ^f	31,199	10	16	9	15 ^f
1950	57,518	22	52	37	34	30,190	16	30	37	17	28,864	11	13	17	13
1951	57,594	30	53	40	43	30,608	18	30	34	23	29,027	18	24	11	22
1952	57,594	18	37	34	27	30,983	21	24	37	17	29,027	10	13	15	10
1953	57,594	24	45	39	34	31,358	14	37	29	30	29,027	6	21	11	19
1954	57,594	20	58	48	39	31,734	15	36	45	24	29,027	3	20	17	17
1955	57,594	25	43	48	32	32,109	9	27	33	20	29,027	8	13	13	13
1956	57,594	25	39	45	31	32,484	11	25	35	18	29,027	6	16	20	19
1957	57,594	20	34	44	23	32,859	17	24	33	18	29,027	4	7	12	6
1958	57,594	17	35	42	24	33,234	15	28	38	23	29,027	9	15	22	12
1959	59,641 ^g	14	28	50 ^g	28 ^g	38,432g	9	27	378	22 ^g	27,250 ^g	2	9	19 ^g	138
1960	57 ,97 3 -	19	30	53	26	37,512	23	22	40	18	25,782	6	9	14	9
1961	57,973	22	31	50	31	37,512	13	29	34	23	25,782	8.	9	23	10
1962	57,915	13	26	61	19	39,074	13	14	45	11	25,782	4	5	20	5
1963	58,537	19	27	48	22	40,211	16	22	46	22	25,782	5	5	19	5
1964	59,578	14	32	55	26	41,655	15	24	36	20	28,166	4	10	22	10
1965	59,048	10	20	60	17	37,634	3	18	40	12	28,166	1	3	28	2
1966	60,969	12	23	71	15	37,612	9	18	40	12	28,340	5	6	25	5
1967	59,659	11	18	59	16	37,861	14	16	48	13	29,473	1	6	20	5
1968	60,062	9	17	82 ^h	17	38,335	13	14	56	14	30,244	4	6	29	6
1969	60,281	19	12	87	10	38,504	13	9	58	10	30,927	7	3	41	3
1970	60,300g	14	10	86g	14 ^g	38,881g	11	13	62 ^g	98	28,3198	6	7	348	8g
1971	58,977	16	23	71	1,9	37,846	12	18	57	14	26,603	3	8	29	7

^aSource: Tennessee Department of Public Health, Annual Bulletin of Vital Statistics, 1929-1971, Nashville, Tenn.

^bResident population after 1933. Recorded location before 1934.

c(1) Stillbirths (fetal deaths). (2) Infant mortality. (3) Cancer deaths. (4) Congenital malformation deaths.

dPopulation totals as recorded in Annual Bulletin of Vital Statistics. Lack of intercensal estimation is obvious for Oak Ridge from 1952-1964, for Anderson from 1952-1961, and for Roune from 1931-1939.

Oak Ridge established but, until 1949, omitted from population totals, although not from mortality counts.

After 1948, 6th revision of International Classification of Diseases, Adapted USHEW-PHS, in effect. Not strictly comparable to earlier data. (ICD 750-776.)

^gTotal population data used in the absence of data for white population alone.

hNote the increase after 1968 due to changing to the 8th revision of International Classification of Diseases, Adapted USHEW-PHS.

Table 3 Rates of Death from Selected Causes in the White Population Proximate to Oak Ridge, 1929-1971ab

		Anderson (Countric			Roane Co	ounty ^c		City	y of Oak		
Year	(1)	(2)	(3)	(4)	(1)	(2)	(3)	(4)	(1) (2)	(3)	(4)
			36.9	22.4	31.1	82.0	• • • • •	26.2				
1929	59.7	62.2			48.7	80.7	52.1	28.6				
1930	37.5	80.0			37.4	55.3	69.5	21.4				
1931	32.7	77.9			33.7	92.4	47.8	33.7				
1932	45.6	72.6			38.2	74.6	56.5	26.8				
1933	44.5	93.2				47.0	47.8	15.7				
1934	57.2	55.5	25.2	28.6	33.0	72.6	86.9	26.5				
1935	37.7	65.3	45.1	14.5	19.5		60.8	30.6	Ň	lo data a	vailable	
1936	36.5	84.6	54.7	34.8	18.7	83.2	78.2	30.6		before	1949	
1937	32.9	64.0	39.5	32.9	20.4	79.9		27.9				
1938	35.1	59.1	68.7	25.9	41.8	80.1	47.8	12.6				
1020	15.1	45.3	58.5	20.8	23.4	43.2	60.8					
1939	23.6	68.6	42.0	44.5	25.4	59.7	90.7	19.4				
1940		73.6	33.5	49.0	14.5	37.8	33.6	16.0				
1941	28.0	54.9	29.1	21.6	12.4	49.6	62.6	16.5				
1942 1943 ^d	3.3 20.1	37.3	31.9	11.5	20.4	40.9	61.9	16.1				
1944	17.1	51.3	40.8	37.6	21.5	67.2	89.9	31.7 26.6				
1945	14.7	45.3	77.8	27.4	27.8	64.2	46.2					
	12.0	32.0	129.0	23.0	25.2	51.6	66.7	28.8				
1946	21.4	32.3	97.1	24.3	26.2	37.5	100.6	26.2				
1947 1948	22.0	31.9	110.8	25.3	18.9	45.0	109.7	29.6			00.0	48.0
1340				83.6 ^e	30.9	52.0	73.7	80.3	10.2	16.4	28.8	45.0
1949	13.3	35.2	65.5	59.2	20.3	38.0	122.6	56.1	13.4	15.8	58.9	75.0
1950	13.4		64.3	73.1	23.1	38.6	111.1	52.2	19.5	26.0	37.9	34.4
1951	17.3		69.5	47.0	25.1	28.6	119.4	54.7	10.6	13.7	51.7	65.6
1952	10.5		59.0		15.6	41.3	92.5	95.8	6.9	24.2	37.9	65.0
1953	14.3	26.7	67.7	58.9				75.7	3.2	21.1	58.6	58.7
1954	10.9	31.7	83.3	67.7	13.9	33.4	141.8	62.3	9.0	14.6	44.8	44.7
1955	14.2		83.3	55.6	9.3	27.9	102.8	55.5	7.8	20.9	66.2	62.9
	15.9		78.1	53.9	11.9	26.9	107.7		6.1	10.6	41.3	20.6
1956			76.4	40.0	19.0		100.4	54.7	14.3	23.8	75.8	42.0
1957		-	72.9	41.6	17.0	31.7	114.3	69.3				47.
1958	11.2				10.3	31.0	96.3 ^f	57.2	3. <i>5</i> °	16.0	69.7 ^f	
1959	10.1		83.8	47.0	27.3		106.6	48.0	10.8	16.1	54.3	34.9
1960	13.7		91.4	44.8			90.6	31.4	14.1	15.8	89.2	38.
1961		2 22.8	86.2	53.4	15.8		115.2	28.2	7.5	9.4	77.6	20.
1962	10.	2 20.3	105.3	32.8	16.0			52.3	9.7	9.7	73.7	19.
1963			82.0	37.6	20.7				7.9	19.6	78.1	35
1964	11.	5 26.2	92.3	43.6	19.				2.4	7.1	99.4	7.
	_			28.8	4.				12.8	15.4	88.2	17
1965				24.6	14.					16.9	67.9	17
1966				26.9	23.					16.6	95.9	19
1961 1961	•			28.3	21.	8 23.5	146.1				132.6	9
	-		_		21.	4 14.8	150.6			8.0	132.6 120.1	-
196						-				19.5		26
197									8.4	22.5	109.0	
197	1 17	.4 24.9	120.4	32.3	10			of Vital S				

^aSource: Tennessee Department of Public Health, Annual Bulletin of Vital Statistics, 1929-1971, Nashville, Tenn.

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^bResident population after 1933. Recorded location before 1934.

c(1) Stillbirths (fetal deaths). (2) Infant mortality. (3) Cancer deaths. (4) Congenital malformation deaths. dOak Ridge established but, until 1949, omitted from population totals, although not from mortality counts.

eAfter 1948, 6th revision of International Classification of Diseases, Adapted USHEW-PHS, in effect. Not strictly comparable to earlier data. (ICD 750-776.)

^fTotal population data used in the absence of data for white population alone.

Table 4 Deaths and Death Rates from Selected Causes in the White Population of the State of Tennessee^a

			De	athsb		Death Ratesb				
Year	White population	(1)	(2)	(3)	(4)	(1)	(2)	(3)	(4)	
1929	2,155,034	1813	2908	1229	1112	42.3	67.9	57.0	26.0	
1930	2,144,781	1827			1192	41.1				
1931	2,169,427	1722	2713	1272	1071	38.4				
1932	2,194,073	1659	2767	1308	1389	37.4	60.5			
1933	2,218,720	1584	2679	1343	1105	37.4	62.3 63.5	59.6 60.5		
1934	2,243,366	1602	2924	1447	1159					
1935	2,268,013	1578	2600	1492	1081	36.3	66.2	64.5	26.2	
1936	2,292,659	1433	2639	1561		35.5	58.4	65.8	24.3	
1937	2,317,306	1460	2371		1077	33.8	62.3	68.1	25.4	
1938	2,341,952	1402	2613	1601 1775	1072 1112	33.6 30.9	54.6 57.6	69.1	24.7	
1939	2,366,599	1288	2157	1706	•			75.8	24.5	
1940	2,413,698	1236	2411		1102	28.7	48.0	72.1	24.5	
1941	2,440,866	1116		1830	1129	25.6	49.8	74.1	23.1	
1942	2,468,038		2482	1908	1188	22.3	49.5	78.2	23.7	
1943	2,495,201	1091 1186	2383 2410	1914 2004	1278 1254	20.1 20.3	43.9	77.6	23.5	
1944	2,522,367						41.3	80.3	21.5	
1945	2,549,536	1077	2412	2095	1276	10.1	42.8	83.1	22.6	
1946	2,576,706	1075	2398	2141	1245	20.1	44.8	84.0	23.3	
1947		1271	2300	2352	1488	19.8	35.8	91.3	23.2	
1948	2,603,876	1344	2451	2426	1624	18.7	34.1	93.2	22.6	
	2,631,041	1247	2393	2549	1622	18.6	35.7	96.9	24.2	
1949 ^c	2,715,653	1240	2554	2732	1696	18.5	38.1	100.6	62.5°	
1950	2,758,918	1130	2186	2837	1472	17.5	33.9			
1951	2,806,084	1172	2095	2994	1562	17.5		102.8	53.4	
1952	2,842,740	1114	2000	2993	1444	17.1	31.3	106.7	55.7	
1953	2,879,409	1008	1784	3097	1346	15.4	30.6 27.3	105.3 107.6	50.8 46.8	
1954	2,916,072	1048	1859	3180	1423	15.5				
1955	2,952,730	998	1728	3228	1311		27.5	109.1	48.8	
1956	2,989,392	1035	1665	3359		15.0	25.9	109.3	44.3	
1957	3,026,051	958	1651	3380	1313	15.7	25.2	112.4	43.8	
1958 ^c	3,062,717	960	1704	3564	1280 1324	14.6 14.8	25.2 26.2	111.7 116.4	42.3	
T959	2,988,879	886	1694	3636					43.2	
1960	2,977,753	923	1610	3628	1369	13.7	26.2	12 1 .7	45.7	
1961	2,977,753	920	1607	3028 3775	1311	14.5	25.3	121.8	43.9	
1962	3,032,532	929	1543		1307	14.4	25.2	126.8	43.9	
1963	3,081,233	884	1459	3810 4187	1264 1196	14.8 14.2	24.5	125.6	41.7	
1964	3,168,049	886	1547				23.5	135.9	38.8	
1965	3,210,400	799	1347	4019	1225	14.2	24.7	126.9	38.7	
1966	3,246,900				1095	14.2	23.5	131.3	34.2	
1967	3,251,200	826	1186	4277	993	15.4	22.1	131.7	30.6	
1968 ^c	3,322,600	728 769	1107 1039	4535 4713	950	13.7	20.9	139.5	29.1	
1969	3,294,331				916	14.6	19.7	141.8	26.4	
1970	3,294,331	716	1031	4704	863	13.0	18.7	141.2	25.8	
1971	3,349,611	765	1061	4964	934	13.5	18.7	150.7	28.4	
	J,J77,011	698	1054	5048	888	12.4	18.8	150.7	26.5	

^aSource: Tennessee Department of Public Health, Annual Bulletin of Vital Statistics, 1929-1971, Nashville, Tenn.

b(1) Stillbirths (fetal deaths). (2) Infant mortality. (3) Cancer deaths. (4) Congenital malformation deaths.

^cNew International Classification of Diseases, Adapted USHEW-PHS, in effect.

d Sixth revision of International Classification of Diseases, Adapted USHEW-PHS, in effect; rate per 10^s population; previously computed per 10^s live births.

More reliable data—age-adjusted cancer mortality from the National Cancer Institute for the years 1950 to 1969 for Anderson and Roane counties—were analyzed statistically to determine if a geographical pattern could be observed.⁴³ The city of Oak Ridge is

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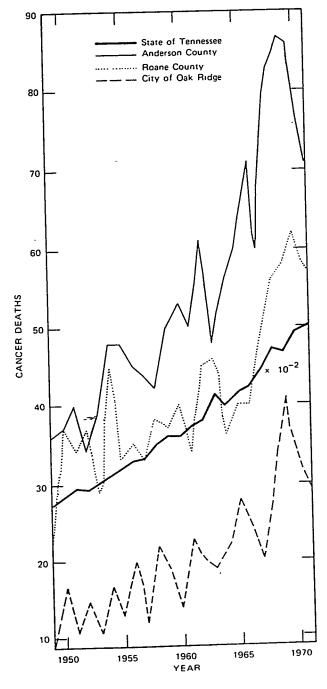


Fig. 2 Cancer deaths in the white population in the state of Tennessee and in the Oak Ridge area, 1949-1971. Source: Ref. 42.

located partly in Anderson County and partly in Roane County; two of the three nuclear facilities are in Roane County, and one is in Anderson County. Figure 3 shows the location of the three facilities and the city of Oak Ridge. Actual deaths from all cancers (including leukemia) were compared with the expected deaths, which were computed using the mortality rate for each cancer and each of four race—sex classes of the population in each county. The results are given in Table 5 (page 661).

The analysis indicates that for every cancer, the number of actual deaths is statistically no different from the number expected for males of both races and for white females. For black females, actual deaths are no different statistically from expected deaths in all cancers examined with two exceptions. For leukemia and lung cancer in nonwhite females in Anderson County, two deaths occurred, whereas only 0.5 would have been expected based on Tennessee rates. (This result is not statistically significant at the 0.05 level using the more appropriate Poisson distribution.) The overall results indicate cancer mortality rates that are not significantly higher than would be expected in the general population. Problems in this analysis include (1) no time trends, (2) migration, and (3) socioeconomic factors, as have been mentioned in regard to previous studies.

CONCLUSIONS AND IMPLICATIONS

Although high levels of radiation are a proven threat to man's health, little evidence has yet been found that low levels of radiation such as might result from the normal operation of nuclear facilities are harmful to the general public. Although much more analysis is needed in this area, existing studies using a variety of methods generally have been unable to detect a rise in measures of ill health in populations living near nuclear facilities. Increases that were observed were either representative of general trends for the state or nation or the continuation of trends existing in the area before the nuclear facility went into operation. The results, although tentative because of the limited scope of research in this area, do suggest by their consistency that no correlation between nuclear facilities and increased mortality in the general public can be substantiated.

Since the National Environmental Policy Act of 1969 considers human health impacts as a major portion of overall environmental impacts, more emphasis should be placed on the inclusion of health and mortality data in environmental impact statements.^{4,4}

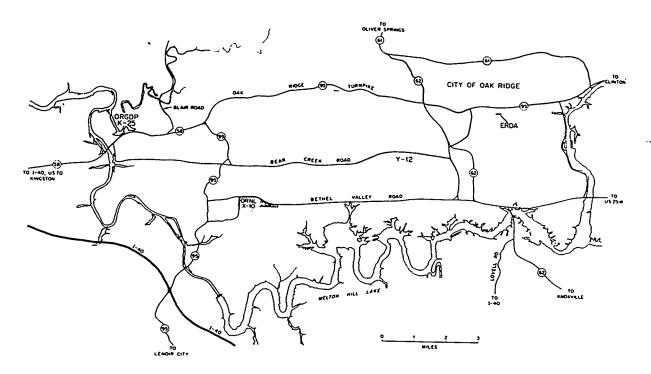


Fig. 3 The city of Oak Ridge, Tenn., and the three nearby nuclear facilities Y-12, X-10, and K-25.

If such data were included, past trends in an area where a power plant, whether nuclear or nonnuclear, is to be located could be used as a baseline against which to measure future changes in health in the area. When widespread utilization of such public health statistics is begun, more meaningful health and mortality statistics will be needed and, hopefully, will be made available. Then trends in various measures of health in areas where power plants are located will allow us to make a more definitive determination of the relative risk of such plants to the public.

Until that time, we must rely on the few studies of public health effects, on results from animal experiments and occupational exposures, and on federal safety regulations as indicators of the safety of power-plant operations to the general public. To date, studies using mortality data from vital statistics have been inadequate for hypothesis testing. These studies should be used solely to indicate the need for more in-depth studies examining hypotheses suggested by trends seen in the vital statistics.

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Table 5 Age-Adjusted Mortality Rates, Actual and Expected Deaths for Selected Cancer Sites by Sex and Race in the State of Tennessee, Anderson County, and Roane County, 1950-1969 (Ref. 43)

				Anderson	County			Roane Co	unty	
Cancer site*	Tenn Observed number	Observed rate	Observed number	Observed rate	Expected number	X2†	Observed number	Observed rate	Expected number	X2†
All cancers WM WF	38,356 35,763	146.3 116.0 163.8	544 510 22	143.8 117.4 236.3	553.5 503.9 15.3	0.16 0.07 2.99	432 384 22	154.4 120.2 145.3	409.3 370.6 24.8	1.25 0.49 0.32 0.60
NM NF	7,874 2,268	142.5	22	213.3	14.7	3.63	26	165.8	22.4	0.00
Leukemia WM WF NM NF	2,268 1,700 301 222	8.4 5.6 6.0 3.9	39 32 1 2	8.7 6.2 9.6 16.7	37.7 28.9 0.6 0.5	0.05 0.33 0.23 5.03‡	20 20 2	6.4 6.0 12.5	26.3 18.7 1.0	1.49 0.10 1.13
Lung WM WF NM NF	8,885 1,673 1,387 300	33.5 5.5 28.8 5.5	151 23 6 2	38.6 6.0 59.0 20.8	131.1 21.1 2.9 0.5	3.04 0.17 3.22 4.09‡	111 15 2 1	38.7 4.7 11.7 6.0	96.1 17.6 4.9 0.9	2.3: 0.3 1.7- 0.0
Bone WM WF NM NF	371 366 59 43	1.4 1.2 1.2 0.8	8 4	· 1.7 · 0.7	6.6 6.9	0.30 1.19	5 6	1.4 1.8	5.0 4.0	0.0 1.0
Thyroid WM WF NM NF	91 195 12 28	0.3 0.6 0.3 0.5	2 2	0.6 0.6	1.0 2.0	1.00 0.0	2	0.6	2.0	0.0

*WM = white male; WF = white female; NM = nonwhite male; NF = nonwhite female.

 $\dagger X^2 = \frac{(observed - expected)^2}{opposted}$, with one degree of freedom, and with expected based on the state rate applied to local expected

population.

 \pm Significant at the 0.05 level (X² > 3.84).

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